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## EDITORIAL NOTES.

The conditions in which icterus is observed are so numerous as to lead to much controversy in regard to its pathogenesis. Apart from those cases due to pure mechanical obstruction, icterus may possibly be due to swelling of the liver cells themselves, causing a narrowing of the finer bile ducts and rendering the passage of abnormally viscid bile quite difficult. In toxæmic cases, this action is quite probable, the increased blood destruction leading to excessive formation and inspissation of the bile.

There is to date no definite confirmation of Minowski's ingenious hypothesis that, under certain conditions, perverted function of the liver cells may bring about the discharge of bile into the lymph and blood, in the complete absence of any obstruction.

We have long had experimental proof that massive destruction of erythrocytes by hemolytic poisons could produce icterus. The much-wished-for clinical proof has been furnished by the work of Chauffard and confirmed by that of Widal, Abrami, Brulé, Oettinger of Paris, Parkes-Weber of England and von Stejskal of Austria (to mention only the pioneers).

It was shown that in a number of cases there exists a marked fragility of the red blood corpuscles on exposure to hypotonic solutions of sodium chlorid. There is also a decrease in the average size of the

red blood corpuscles, and on vital staining, peculiar basophilic granulations of the erythrocytes are seen, their occurrence being interpreted as an indication of active blood regeneration.

"Hemolytic jaundice" is now accepted by most clinicians as a distinct clinical entity, and is here considered as such.

A number of the patients are congenitally icterics, and the disease sometimes occurs in families. Jaundice may come on immediately after birth or not until puberty. There is always a moderate anemia, in spite of which subjective symptoms are usually absent. Icterus is usually not intense; there are no signs of obstruction of the bile ducts, and symptoms of cholemia, such as bradycardia, pruritus, xanthomas and hemorrhages are likewise absent in spite of the presence of bile pigment (but not of urobilin) in the blood. The stools are highly colored, the urine contains no bile. The spleen is practically always enlarged in the congenital cases. It is probable that some so-called splenic anemias are really instances of this disease.

In the acquired hemolytic icterus the anemia is far more intense, and, curiously enough, the corpuscular fragility is not so marked as in the congenital type. In addition, an auto-agglutinative power of the serum is at times observed. The most important forms of the acquired type may simulate (1) cholelithiasis, (2) pernicious anemia with jaundice, (3) chronic infectious cholangitis, (4) splenic anemia or (5) icterus gravis. (The recognition of the acquired types is particularly important, because some of them can be greatly improved if not cured by the persistent administration of iron.)

It is impossible to say where the hemolysis occurs; some insist that it is in the spleen (and report cases cured by splenectomy), others that it is in the blood. At any rate, the important problem as to the primary cause of the condition is certainly at present impossible of solution.

But little attention has been paid to this subject in American literature. It is hoped that Thayer's review in the Johns Hopkins Bulletin will be consulted by those encountering similar cases. The laboratory tests for corpuscular fragility are a trifle tedious, but not at all difficult of execution in hospital, as the writer can testify. René Bine.

Some of us will never cease to stand aghast at the ease with which anxious families are placated with polysyllabic reverberations. This pregnable quality of human nature, the awe of the unknown, is seized upon by many a practitioner of many patients and fewer morals,